Title: EFFECT OF CARDIAC TAMPONADE ON LEFT VENTRICULAR CONTRACTILITY

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Introduction. Acute cardiac tamponade represents a serious complication following open heart surgery. Whether tamponade directly causes left ventricular (LV) ischemia due to lowered arterial pressure and elevated intracardiac pressures remains controversial. Depression of LV contractility, potentially secondary to ischemia, and myocardial necrosis have been reported following tamponade[1-3]. However, on the basis of an earlier study showing normal myocardial metabolism during severe tamponade[4], we questioned whether myocardial contractility is depressed during tamponade. One problem when interpreting earlier studies is that conventional measurements of LV performance are substantially influenced by changes in loading conditions such as preload and afterload. The slope of the LV end-systolic pressure-volume relation (termed E_{ES}) represents a load-insensitive measure of LV performance. Therefore, we assessed LV contractility by determining E_{ES} during graded levels of experimental cardiac tamponade.

Methods. In 8 open-chest anesthetized dogs, a transducer-tipped catheter and a 7 French octapolar impedance catheter connected to a signal conditioner-processor (Leycom Sigma-5, Oestgeest, The Netherlands) were inserted into the LV to provide a continuous assessment of LV pressure and volume, respectively. End-systolic pressure-volume loops were recorded during acute preload reduction from bicaval occlusion. E_{ES} was determined from linear regression of the end-systolic pressure and volume points. Through a catheter secured in the pericardial space, 30 ml of warm sucrose solution (290-310 mosm/L) were infused to permit accurate assessment of pericardial pressure by a fluid-filled catheter. After baseline measurements were obtained, more sucrose solution was infused to reduce mean arterial pressure (MAP) to 90, 75, and 60 mmHg.

Data, expressed as mean $\pm$ SEM, were analyzed by analysis of variance for repeated measures with a p value < 0.05 considered significant. This study was approved by the Institutional Research Practice Committee.

Results. Incremental infusions of sucrose significantly increased pericardial pressure from 3 $\pm$ 1 mmHg to 6 $\pm$ 1, 8 $\pm$ 1, and 9 $\pm$ 1 mmHg, respectively. Accordingly, MAP significantly decreased from 105 $\pm$ 3 mmHg to 89 $\pm$ 2, 75 $\pm$ 2, and 59 $\pm$ 1 mmHg, respectively. The end-systolic pressure-volume relations were well-approximated by straight lines having correlation coefficients of 0.96, 0.98, 0.96, and 0.98, respectively. E_{ES} was 6.3 $\pm$ 1.2 mmHg/ml at baseline and 7.7 $\pm$ 1.8, 8.5 $\pm$ 1.3, and 9.2 $\pm$ 1.5 mmHg/ml during mild, moderate, and severe tamponade. Although the mean value of E_{ES} increased with each successive level of tamponade (Figure 1), the change did not reach statistical significance.

Discussion. These results show that LV contractility is not reduced during severe cardiac tamponade. Furthermore, our E_{ES} data suggest that tamponade to a MAP of 60 mmHg does not induce myocardial ischemia since ischemia per se reduces LV contractile state and E_{ES}. Below this range, hypoperfusion of all vascular beds, including the coronary arteries,[3] would be expected. The clinical implication is that hemodynamic problems with cardiac tamponade are related to impaired ventricular filling and not to myocardial ischemia with depressed LV contractility.

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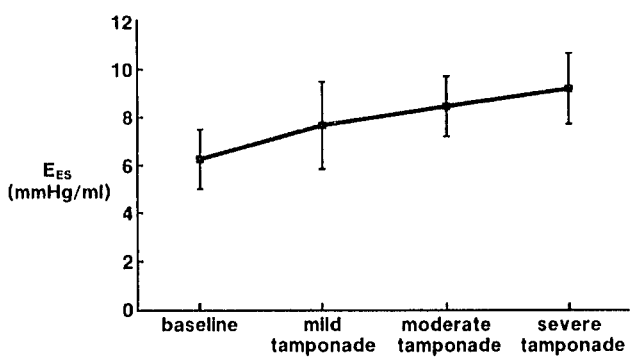

Figure 1. Effect from progressive levels of cardiac tamponade on E_{ES}.